DEMOGRAPHIC, CLINICAL, INVESTIGATIONAL AND ETIOLOGICAL PROFILE OF PATIENTS WITH CEREBRAL VENOUS THROMBOSIS AND ANALYSIS OF SHORT TERM OUTCOME

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BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **"DEMOGRAPHIC, CLINICAL, INVESTIGATIONAL AND ETIOLOGICAL PROFILE OF PATIENTS WITH CEREBRAL VENOUS THROMBOSIS AND ANALYSIS OF SHORT TERM OUTCOME"** is a bonafide record work done by **Dr. R.BALAKRISHNAN** under my direct supervision and guidance, submitted to the Tamil Nadu Dr. M.G.R. Medical University in partial fulfillment of University regulation for DM, Branch I –Neurology.

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DECLARATION

I **Dr. R. BALAKRISHNAN** solemnly declare that the dissertation titled **"DEMOGRAPHIC, CLINICAL, INVESTIGATIONAL AND ETIOLOGICAL PROFILE OF PATIENTS WITH CEREBRAL VENOUS THROMBOSIS AND ANALYSIS OF SHORT TERM OUTCOME"** has been prepared by me. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, diploma to any other University board either in India or abroad.

This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulation for the award of D.M. degree Branch – I (Neurology) to be held in August 2009.

Place: Madurai **Dr. R. BALAKRISHNAN**

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CONTENTS

INTRODUCTION

Cerebral venous / sinus thrombosis has been recognized since the early 19th century, but still remains a diagnostic and therapeutic challenge. Patients with cerebral venous thrombosis usually presents with headache, seizures, papilledema, altered sensorium and focal deficits due to thrombosis of intracranial veins / sinuses resulting in hemorrhagic infarction and increased intracranial tension. The above features are present in various combinations ranging from syndrome of raised intracranial pressure without localization to deep altered sensorium and dense hemiparesis. Cerebral venous thrombosis forms a distinctive subgroup of cerebrovascular disease in India and is a leading cause of mortality in women of reproductive age group. In India, most of the cases are seen in postpartum period in women, while alcoholism is a significant risk factor in males. The high incidence of postpartum cerebral venous thrombosis is due to lack of proper hygiene resulting in high incidence of puerperal sepsis, high prevalence of anemia, and restriction of water intake during labour as a cultural practice. Other predisposing and causative factors for cerebral venous thrombosis are oral contraceptive pills intake, procoagulant states, internal malignancies, infections. Cross et[4] al noted "usually recovery is rapid and complete if the patient survives

the acute episode". ¾ cases of cerebral venous thrombosis in pregnancy and puerperium reported by him survived with good recovery. However in pre imaging era, cerebral venous thrombosis has been diagnosed exclusively at autopsy and therefore thought to be always lethal. After the introduction of heparin in treatment of cerebral venous thrombosis, mortality has come down significantly and most of the recent studies reporting mortality of < 20% compared to earlier studies which reported mortality between 30 - 50%. However the outcome of cerebral venous thrombosis is highly unpredictable and it is not unusual to see dramatic recovery in deeply comatose patients and sudden worsening of conscious patient due to extension of thrombus. With the advent of CT and MRI / MRV the diagnosis of cerebral venous thrombosis has improved significantly. CT scan commonly shows hemorrhagic infarction with or without "cord" / "empty delta sign". MRI / MRV diagnose & confirm the diagnosis of cerebral venous thrombosis even if the CT scan is normal and also diagnose the thrombosis of cortical veins and deep veins. After introduction of MRI many of the cases earlier diagnosed as idiopathic intracranial hypertension have been noted to have sinus thrombosis. Involvement of superior sagital sinus of varying extend with or without involvement of transverse sinus and sigmoid sinus with thrombosis of cortical veins has been reported. Involvement of deep venous system is less common than the superficial venous system but by means not rare. Due to

multifactorial causation of this condition, and puerperal cerebral venous thrombosis is the major group among cerebral venous thrombosis seen in Indian series it is worthwhile to study the demographic, clinical, investigational and etiological profile of patients with cerebral venous thrombosis and worthwhile to compare demography, clinical, radiological features and analysis of short term outcome between puerperal, non puerperal female and male patients with cerebral venous thrombosis

REVIEW OF LITERATURE

Cerebral venous thrombosis or sinus venous thrombosis, as the name implies is a condition which involves cerebral venous sinuses and veins together or independent of each other with thrombotic event of varied temporal evolution. The clinical presentation is varied ranging from syndrome of raised ICT without localization to seizures, focal deficits & deep altered sensorium[2,3]. Some patients even present as behavioral disturbances as the predominant clinical manifestation, confusing the picture with postpartum psychosis. Strokes resulting from cerebral venous thrombosis usually affect young persons particularly women in reproductive age group & carry high mortality if not managed adequately. The term primary or idiopathic CVT is used when no specific etiological factor is evident. Secondary sino venous thrombosis results from a variety of causes that include injury, infection, hematological disturbances, dehydration etc. In India, CVT in early puerperal period is very common i.e. 10 – 12 times higher than in the

west[12].

Historical background:

The wide spectrum of clinical feature in CVT, the varied & changing etiological factors & the apparent rarity of the condition had made advance in knowledge slow & uneven. Periods of relative neglect had been interspersed with burst of enthusiastic discussion. The earliest reference to CVT was that of Ribes in 1823[1]. He described in detail the clinical & post partum findings of 45 year old man who had thrombosis of superior sagital and lateral sinuses, subdural effusion and metastatic carcinoma in the brain. The first case of puerperal venous thrombosis was reported by John Abercromobie in 1828. His patient, a 21 year old woman, developed headache, delirium and initially right sided then generalized seizures at the beginning of second week after delivary. Autopsy showed ischemic and haemorrhagic infarcts with thrombosed and sclerosed cortical veins. Quinke and Nonne identified the clinical syndrome of pseudo tumor cerebri (a term coined later) as a clinical counter part to sinus thrombosis. In 1940, Martin and then Martin and Sheehan focusing on postpartum sinovenous thrombosis evoked a number explanation including air embolism, embolism by way of paraveretebral venous plexus and damage to venous walls, secondary to venous engorgement of pregnancy. Kalbag

and Woolf, Sir Charles Symonds and others gave a precise clinical description of CVT after 1940. After introduction of CT scan and later with the use of MRI with MRV, diagnosis of CVT has become simpler as these imaging modalities are quite sensitive in detecting CVT. Several large series with confirmation of diagnosis by angiograms, surgical exploration, and autopsy and recently with CT and MRI studies have been reported from Indian subcontinent.

Epidemiology:

The true incidence of CVT is unknown. Ehlers and Courville[14] found only 16 superior sagital sinus thrombosis in a series of 12,500 autopsies (0.12%). Towbin found CVT in 9 % of 182 consecutive autopsies. However, with the more recent reports of large clinical series, the true incidence of CVT is probably higher than that derived from autopsy figures. Exact figures however remain elusive. People of all age groups may be affected by CVT but there is preponderance in young women because of specific causes like use of oral contraceptives, pregnancy and puerperium. Puerperal CVT has been reported to account for up to 15 – 20 % of "young stroke". It is the commonest cause of stroke in young women in India. 50% of strokes in Indian women are related to pregnancy and puerperium and 95.5% of these are due to CVT. In western countries, the incidence of CVT related to pregnancy and puerperium ranges from 1 in 1666 – 10,000 pregnancies. CVT has been reported to occur in 4.5/1000 obstetric admissions in India.

Relevant venous anatomy:

The cerebral venous system comprises of cerebral veins that empty into dural sinuses which then drain the blood into two internal Jugular veins. Embryologically, the entire cephalic drainage may be subdivided into an outer and superficial segment, which drains the scalp, underlying muscle and tendons, an intermediate segment which drains the skull, diploe and dura matter, and a cerebral segment, consisting of the veins that drain the brain. The cerebral segment may be further subdivided into a superficial cerebral group of veins and a deep cerebral group of veins. The superficial cerebral veins coalesce on the Pial surface draining out the blood from the outer 1 or 2 cm of cortex and the underlying white matter. Venous blood in these vessels travels in a centrifugal direction and ultimately terminates in one of the dural sinuses. The deep cerebral veins serve to drain blood in a centripetal direction away from deep white matter, the basal ganglia, and the diencephalons. Tributaries draining many of the deep structures of the cerebrum join veins in the lateral angles of the ventricles and form a sub ependymal plexus. The veins of this plexus empty into the internal cerebral veins, which join the great cerebral vein of Galen.

Superior sagital sinus (SSS)

SSS lies in the attached border of the falx cerebri and runs from the

foramen caecum to the occipital protuberance, where it joins straight sinus, lateral sinus and torcular herophilli i.e confluence of sinuses. The anterior part is narrow or sometimes absent or replaced by two superior cerebral veins that join behind the coronal suture. Consequently anterior part of sinus is often poorly visualized on angiography and in MRV and its isolated lack of filling is not sufficient to indicate thrombosis. The SSS receives superficial cerebral veins and drains major part of cortex. It also receives diploic veins, themselves connected to scalp veins by emissary veins, which explains the incidence of SSS thrombosis after cutaneous infections and contusions. SSS and other sinuses play a major role in CSF circulation because they contain most of the arachnid villi & granulations in which most of the CSF absorption takes place. Thus there is a direct dependency of CSF pressure accounting for the frequency of raised intra cranial pressure in SSS (or) lateral sinus (LS) thrombosis.

Lateral Sinus (LS)

The lateral sinus starts from torcular herophilli to the jugular bulb and consist of transverse & sigmoid portions. They drain blood from cerebellum, brain stem & posterior portion of cerebral hemispheres. They also receive some veins from middle ear, another possible source of septic thrombosis. Numerous LS anatomic variations may be misinterpreted in sinus occlusion on angiography. The right LS is more of the direct continuation of SSS & frequently larger than left LS

which receives most of its blood from straight sinus. In Hackers study transverse portions were not visualized on ipsilateral carotid angiogram in 14% cases on left side and 33% on right side, where as sigmoid portions, which may be directly injected via cerebral veins failed to fill in 4% of cases on left side and was always demonstrated on the right side. An isolated lack of filling of a left transverse sinus is more suggestive of hypoplasia than of thrombosis.

Cavernous sinus

This sinus drains venous blood from the orbit through ophthalmic veins and from anterior part of the base of the brain via spheno palatine sinus and middle cerebral veins. They empty into both superior & inferior petrosal sinuses and ultimately into internal jugular veins. Because of their situation cavernous sinuses are often thrombosed in relation to infections of the face or sphenoid sinusitis. In contrast to other sinuses, infection is the leading cause of cavernous sinus thrombosis.

Cerebral veins:

They can be roughly divided into 3 groups.

1. Superficial cerebral veins.
2. Deep cerebral veins.
3. Veins of posterior fossa.

Superficial cerebral veins

Some of the cortical veins, the frontal parietal, occipital and superior cerebral veins drain the cortex ascending to SSS while others mainly the middle cerebral veins drain into the cavernous sinus. Trolard's great anastamotic vein connects the SS to middle cerebral veins, which are then connected to LS by vein of Labbe´. The cortical veins present some peculiarities that are important to know to understand some of the clinical features of CVT. They have thin walls, no muscle fibers and no valves. These features allow for dilatation and reversal of blood flow when the sinus into which they drain are occluded. They are linked by various anastamosis, allowing development of collateral circulation (angiographically visible as corkscrew vesselS). This probably explains the good prognosis of some of the venous thrombosis.

Deep cerebral veins

The internal cerebral and basal veins joins to form great vein of Galen , which continues as straight sinus drain blood from deep white matter of the cerebral hemispheres and from basal ganglia . In contrast to superficial system the deep system anatomy is always visualized on angiography, so that thrombosis is easily recognized.

Veins of posterior fossa: These are 3 groups.

1. Superior veins draining into Galenic system.
2. Anterior veins draining into petrosal sinuses.
3. Posterior veins draining into the torcular or neighboring SS and LS.

They are variable in course and angiographic diagnosis of their occlusion is extremely difficult.

Microscopic Anatomy of Cerebral Veins and Sinuses.

Capillaries open into cerebral venules which apart from their wider lumen are indistinguishable from them. These venules join the small medullary or cortical veins. These vessels reach the ventricular or cortical surfaces, either directly or indirectly, after fusion with neighboring veins forming larger vascular stems. The walls of cerebral veins consist of an endothelial lined tunica intima. Surrounding the endothelium is a thin adventitial layer. Veins do not have clearly defined muscular layer or valves. There is little to suggest that the veins receive vasomotor innervation. As these vessels approach their destination they become more fibrous and resemble closely the structure of dural sinus. The strategic location of the sinuses within the major folds or junctions of dura, the firm attachment of the dura to base, and the tough fibrous consistency maintains the patency of the sinuses all the time. The walls of dural venous sinuses consist of an inner lining of endothelium and an outer layer essentially the same as dura

elsewhere. The outer layer consists chiefly of fibroblasts and large interlaced bundles of collagenous fibres. A few nerve fibres, presumably afferent have been reported to innervate along the dural venous sinuses. The walls of the venous sinuses are not uniformly smooth and in certain locations like middle third of SSS area thrown into folds or membranous irregularities. According to one hypothesis, these folds may perform valve like action, while other authors suggested that they may be important in maintaining laminar flow.

Causes of CVT:

Several medical, surgical and gynaeco - obstetric ailments as well as a number of regional causes like infective, trauma, tumors etc. have been implicated in the causation and predisposition to CVT. Table (1) lists the recognized causes or predisposing conditions.

Table 1: causes of cerebral venous thrombosis.

A. SEPTIC DURAL VENOUS THROMBOSIS.

Local: Sepsis, trauma

Intra cranial infections, abscess, empyema and meningitis

Otitis media, Sinusitis

Tonsilitis, Stomatitis.

Systemic: Bacterial (Typhoid, TB, Septicemia, Endocarditis)

Viral (Measles virus, Hepatitis viruses, Herpes simplex

Viruses, HIV, Cytomegalo viruses)

Parasitic (Malaria, trichinosis)

Fungal (Aspergillosis).

B. NONSEPTIC DURAL SINUS THROMBOSIS.

Altered Hemodynamic states

Dehydration

Fever

Cardiac failure.

Hematological disorders

Polycythemia vera

Secondary polycythemia

Disseminated intravascular coagulation

Thrombotic thrombocytopenic purpura

Heparin-induced thrombocytopenia

Sickle cell anemia

Paroxysmal Nocturnal hemoglobinuria

Thrombocytosis

Leukemia

Severe anemia

Anti Thrombin –III Deficiency

Protein C & S deficiency

Factor V Leiden

Antiphospholipid antibody syndrome

Hormonal dysfunction

Oral contraceptive use

Pregnancy and puerperium

Androgens

Trauma

Penetrating & nonpenetrating head injuries

Surgery

Cardiac pacemakers

Jugular Venous Catheters

Metabolic disorders

Homocystinuria

Osteopetrosis

Diabetes mellitus

Neoplasia

Meningioma

Metastasis

Inflammatory disorders

Behcet's disease

Sarcoidosis

SLE

Wegners Garnulomatosis

Polyarteritis nodosa

Inflammatory bowel disease

Ulcerative colitis

Crohn's disease

Vascular disoders

Atrerio venous malformation

Arterial occlusions

Sturge weber syndrome

CLINICAL PROFILE:

CVT may occur at anytime from infancy to old age. The spectrum of symptoms and signs among patients with CVT is remarkably variable. Patients present with varying combinations of headache, seizures, aphasia, behavioral abnormalities, altered sensorium and focal deficits. The onset may be acute (<2 days, 20-30%), sub acute (2 days to 30 days, 50-60%) and chronic (>30 days, 10-20%). The presentation is acute in obstetric and infectious CVT while a slowly progressive disease is more common in inflammatory and idiopathic cases.

Bilateral papilledema and symptoms of raised intracranial pressure occur in those with large sinus (SSS and LS) thrombosis blocking CSF absorption. Cortical deficit like agnosia, apraxia, cortical blindness and aphasia do occur but better recognized in mild illness with good sensorium. Some patients may present with psychotic features before manifestations of increased ICT / or focal deficits sets in. Monoplegia (brachial / crural) or Hemi paresis with leg more affected than arm, intact language despite right hemiparesis are all common but generally regress without residual deficits. Cranial nerve palsies are reported in 12% of all cases of cerebral venous thrombosis. The cranial nerves that have been described to be involved are III, IV, V, VI, VII, VIII, IX, X and XI, and the involvement can be multiple or single. In rare cases, cranial nerve palsies can be the only sign of cerebral venous thrombosis, especially when there is the involvement of the transverse/sigmoid sinus (VI, VII and VIII cranial nerves). Along with other neurological events, involvement of cranial nerves is known from the previous literature; VI nerve palsy in one patient with lateral sinus thrombosis, diplopia due to VI nerve palsy and signs of V nerve irritation with temporal and retro-orbital pain, it has also long been known as the Gradenigo syndrome, suggesting involvement of the nerves at the petrous apex. The unilateral or bilateral VI cranial nerve involvement can also be due to the intracranial hypertension itself. The involvement of the III, IV, V and VI cranial nerves can be due to the thrombosis of

the anterior cavernous sinus. An involvement of the IX, X and XI cranial nerves is possible when the location of the thrombosis is in the posterior cavernous sinus or the internal jugular vein, or the deep venous system or the cerebellar veins.

Cerebellar infarcts with edema acting like space occupying lesion, requiring surgical decompression are rarely encountered. Neuropsychological deficits such as impaired anterograde memory, dementia, akinetic mutism, and abnormal movements such as athetoid movements and dystonia, can in rare cases be symptoms of deep cerebral venous thrombosis.

A generalized encephalopathic illness without localizing signs or recognizable features of raised intracranial pressure is another pattern of presentation .A depressed level of consciousness is the most constant finding, varying from drowsiness to deep coma.

Subarachnoid hemorrhage has been described as the initial presentation of dural sinus thrombosis. The exact cause of subarachnoid hemorrhage associated with cerebral venous thrombosis is unknown. Isolated psychiatric symptoms such as irritability, anxiety, depression, psychosis, delirium and amnesia are known to be the prevailing symptoms of cerebral venous thrombosis. They can be misleading in the postpartum period.

Reversible parkinsonism and MRI diffusion abnormalities have been described as a presenting symptom of cortical venous thrombosis. Finally, cerebral

venous thrombosis can also be asymptomatic, particularly in the case of lateral sinus thrombosis, which can be observed on a routine CT.

The following table shows the clinical features of CVT in various Indian studies.

Table – 2

Clinical features of CVT in various Indian series

No.	Clinical Features	Bansal etal[17] 1980 N =138 (%)	Srinivasan[12] 1983 N =135(%)	Nagaraja[3] N 405(%)
1.	Fever	62	16	-
2.	Head ache	48	24	70.8
3.	Vomiting	36	24	38
4.	Seizures			
	GTCS	29	50	39.7
	Focal	17	22	30.1
5.	Dysphasia	25	5	-
6.	Diplopia	1	-	-
7.	Nuchal rigidity	3	10	13
8	Deep leg vein thrombosis	10	-	-
9	Altered sensorium	41	43	58
10	Papilledema	35	16	18.5
11	Ocular palsy	-	2	11
12	Motor deficit	69	49	66.4

In one study by Ameri et al in 1992 showed the most common presenting

symptoms and signs were headache, papilledema, seizures and motor and sensory deficits. The following table shows the clinical features in Ameri series (1992).

Table – 3

Clinical features described by Ameri et al 1992 [18]

No.	Feature	No	Percentage
1.	Head ache	83	75%
2.	Papilledema	54	49%
3.	Motor / sensory deficit	38	34%
4	Seizures	41	37%
5	Drowsiness, mental changes contusion or coma	33	30%
6	Dysphasia	13	12%
7.	Multiple cranial or palsies	13	12%
8.	Cerebellar incoordination	3	3%
9.	Nystagmus	2	2%
10	Hearing loss	2	2%
11.	B/L or alternating cortical signs	3	3%

The setting of puerperal CVT is quiet characteristic. A young multiparus women presents with headache, focal or multifocal seizures, fluctuating neurological deficits and declining sensorium during be second or 3rd week after a full term normal delivery. Seizures are usually seen in clusters or status but are not very difficult to control with usual anti convulsants.

Table – 4

Timing of onset of symptoms to post partum period to various

series

Post Partum day	Bansal et al[17] 1980 N =138(%)	Nagpal 1983 N =34(%)	Srinivasan[12] 1983 N=135(%)	Nagaraja [3] 1987 N= 200(%)
0 – 7 days	52	65	Maximum in first 10 days	30
7 – 14 days	32	64.5	Mostly within 2 weeks	40
14 days – 3 months	16	0	15	-
> 3 months	0	10	0	-
Following abortion	-	3.0	-	34

The rest developed CVT during pregnancy following oral contraceptives. In children and elderly, CVT may present with lethargy and stupor with headache without any focal deficits. Nagaraja et al grouped clinical features of CVT in four categories depending upon the topographical venous involvement.

1. Presentation with seizures, focal deficits and progressively deteriorating consciousness. Thrombosis involves the dural sinuses as well as cortical veins producing cerebral infarction. Seizures may be focal, multi focal or generalized. Paralysis may be unilateral or bilateral and is usually maximal in lower limbs. Later during the course patient may manifest signs of cortical or cerebral herniation leading to coma and death.
2. Presentation with symptoms and signs of raised intra cranial tension

namely headache, vomiting and papilledema. If thrombosis continues to dural sinuses, the course is usually slow and prognosis is favorable.

3. Occasionally thrombosis predominantly involves cortical veins and patient may present with features of space occupying lesion.
4. Rarely, thrombosis predominantly involves the deep venous system. Patient manifest symptoms of raised intracranial tension, focal deficits. Choreoathetosis, ocular signs and coma. If runs a fulminant course.

Cavernous sinus thrombosis is usually due to spread of infection from face, para nasal sinus or intra cranial venous sinuses. It has a distinctive clinical picture where patient presents with fever, chills, toxemia with proptosis, chemosis and painful ophthalmoplegia, initially unilateral but often becoming bilateral. Papilledema and retinal hemorrhage indicate retinal vein thrombosis.

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INVESTIGATIONS:

Computed tomography scan with contrast injection is the first Neuro imaging examination to be carried out when CVT is suspected as it is easily available and has good sensitivity and specificity. Plain CT scan helps to rule out other pathologies such as tumors. CT scan is initially abnormal in 81- 89% of cases of CVT. Normal scans are particularly common early in the course. Some

studies show normal scans up to 25- 40%. CT signs of sinus thrombosis may be focal or generalized and may result from the thrombosis it self or form its sequelae. They are seen before or after the infusion of contrast. Signs may be direct (evidence of thrombosis) or indirect sequelae to thrombosis). On plain CT scan, the thrombosed SSS may appear as an unusually dense triangle which is sometimes referred as “dense triangle sign” (Filled triangle sign) may be seen 60% of patients. Straight sinus and vein of Galen may also appear hyperdense before contrast when they are thromosed. The “cord sign” which is pathognomonic of cortical vein thrombosis, is a round hyper density seen in several sequential slices due to the presence of thrombosis in the lumen of the vein. It is present in 2-25% of patients. Intra parenchymal linear hyper intensities representing thrombosed intra cerebral veins have the same significance. Indirect signs include areas of ischemia may be imaged as mixed density lesion representing hemorrhagic infarction. Hematoma may also be seen. The location of the lesion correlates well with the site of occluded vein. Non hemorrhagic venous infarcts are seen as hypodense areas which are not corresponding to arterial territories. Evidence of increased intracranial pressure such as cerebral edema, compression of ventricles (slit like ventricles), compression of sub arachnoid space may be seen.

The best known and most specific sign seen after contrast is “empty delta sign”[20,38]. This sign consists of a central lucency within the SSS or straight sinus

surrounded by a margin of contrast enhancement. Enhancement of the falx, tentorium can be reliably imaged on contrast CT scan. Isolated gyral or linear enhancement is seen may be interpreted as SAH.

In one Indian study by Dr.Nagaraja et al in 1989 in a study of 69 patients showed normal CT in around 10 % of patients.

Table - 5

CT features of puerperal CVT N = 68 Nagaraja et al 1989[2]

Non contrast Seen	N	%
Cord sign	14	21.9
Diffuse / focal edema	16	25.0
Hemorrhagic lesion	26	40.9
Unilateral	22	
Bilateral	4	
Non Hemorrhagic lesion	33	51.6
Unilateral	16	
Bilateral	17	
Hematoma	2	3.1
Normal	7	10.9
Entered scan : N : 25		
General enhancement	15	60
Empty triangle sign	8	32
Tentorial enhancement	4	16
Normal	1	4

MRI has become the imaging modality of choice for the diagnosis of suspected CVT. It is more sensitive than CT to parenchymal abnormalities, petectial haemorrhage, thrombus formation and blood flow. MRI is superior than CT in providing definitive evidence for CVT. MRI findings depend on the sequence used and stage (age) of thrombosis. In standard spin Echo T_1 and T_2 weighted MRI the main direct sign of CVT is the lack of expected signal flow void. Alteration in blood flow hemoglobin degradation products in thrombosed veins produce signal changes in T_1W, & T_2W images suggest CVT. At very early acute stage (day 1-5) there is absence of flow void and the thrombus appear isointense on T1 and hypo intense on T2W images due to the presence of oxyhemoglobin in the intact red blood cells. This MRI pattern is rarely seen due to the usual delays in presentation and performing MRI examination.

At the sub acute stage (day 6-21), the thrombosis becomes hyperintense, initially on T_1WI (day 6-9) then on T_2 WI (day 10-15) due to conversion of oxyhemoglobin to methemoglobin. Absence of flow void persists. Increased signal intensities on both T_1WI and T_2WI images is be most frequent MRI finding in patients with CVT. This pattern lasts until (21-35 days) after the onset of thrombosis.

At the chronic stage, (>21-35 days) the MRI pattern is more variable. The thrombosed sinus can either remain totally or partially occluded or can recanalize.

In most patients, the chronic thrombus appears heterogenous becoming progressively isointense on T_1W images, and isointense to hyperintense on T_2W images. These findings can last for years and can be mistaken for recurrent CVT.

Gadolinium administration can increase be sensitivity of the plain MRI. MRI is also sensitive for identifying the changes in the mastoid air cells. Echo planar T_2 weighted MRI / Diffusion weighted MRI is also useful in diagnosis of CVT.

Magnetic resonance venography has become the imaging modality most widely used to establish the diagnosis of CVT. MRI can be performed with time of flight (TOF) or phase contrast techniques.

Most imaging protocols utilize TOF technique due to its short acquisition time. Absence of signal within a sinus and its non opacification suggest intra luminal thrombosis. Occluding thrombosis often appear hyper intense. MRV has limitation with false positive / false negative diagnosis. Non dominant (hypoplastic) transverse sinus can be seen in up to 30% normal individual when using TOF MRV leading to erroneous diagnosis of CVT.

Cerebral CT venography, recently dynamic spiral CT techniques have been utilized to study cerebral venous circulation. Can be performed easily and rapidly in acute setting. Filling defects with in the affected sinuses, sinus wall enhancement and abnormal venous collaterals are the usual finding.

Conventional four vessel angiogram allows visualization of be entire venous phase. In recent years, its utility has declined because of its invasive nature and greater availability of MRI / MRV and CT / CTV techniques.

Transcranial Doppler techniques may have a role in diagnosing CVT. Thrombosis of SSS / deep basal vein of Galen, Labbé or Rosenthal can be associated with increased velocities in deep venous system. TCD evaluation may be useful in monitoring changes in venous flow and response to treatment. Available data suggest the value of TCD in CVT is limited.

TREATMENT

Available treatment data from controlled trials favor the use of anticoagulants in patients with CVT because it may reduce the risk of a fatal outcome and severe disability and does not promote ICH. In one prospective study by Einhäupl et al[28] which compared dose adjusted intravenous heparin with placebo in 20 patients, 8 patients in the heparin group recovered completely and none died, whereas only 1 patient in the placebo group recovered fully and 3 patients died. Three patients with previous ICH recovered completely and no new hemorrhages occurred in the heparin group, whereas in the placebo group 2 patients with pretreatment ICH died and 2 new hemorrhages were observed.

The only other randomized trial compared body weight-adjusted subcutaneous low molecular weight heparin (LMWH) with placebo in 60 patients

with CVT. A poor outcome defined as death or Barthel index < 15 was observed after 3 weeks in 6 of the 30 patients treated with LMWH (20%) compared to 7 of the 29 controls (24%). After 12 weeks, 3 patients (10%) in the LMWH group and 6 patients (21%) in the placebo group had a poor outcome, which corresponds to a non significant absolute risk reduction of 11% in favor of the active treatment. No new ICH or secondary worsening of the 15 patients with pretreatment hemorrhage was observed in the LMWH group. A Meta analysis of these two trials showed that the use of anticoagulation led to an absolute risk reduction in death or dependency of 13% with a relative risk reduction of 54%. Although this difference is not statistically significant, both trials show a consistent and clinically meaningful trend in favor of anticoagulants and demonstrate the safety of anticoagulant therapy. However, it is unclear, whether treatment with full-dose intravenous heparin or subcutaneously applied LMWH is equally effective.

There is currently no evidence from randomized controlled trials about the efficacy and safety of either systemic or local thrombolytic therapy in patients with CVT. Thrombolytic therapy has the potential to provide faster restitution of venous outflow and positive effects of local thrombolytic treatment of CVT have increasingly been reported from uncontrolled series. Patients were either treated with heparin and urokinase or heparin and recombinant tissue plasminogen activator (rtPA) which may carry less bleeding complications due to its clot

selectiveness and shorter half-life. Two uncontrolled studies which used rtPA in combination with dose-adjusted intravenous heparin included a total of 21 patients. Both studies showed recanalization was rapidly achieved local thrombolysis may carry a higher risk of bleeding complications compared to anticoagulation particularly if pretreatment ICH is present. Currently, local thrombolysis may be a therapeutic option for patients at high risk for a poor outcome despite heparin therapy. The International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT) identified comatose patients may define a subgroup of patients with CVT who are at high risk of death despite anticoagulation who may be benefited from thrombolysis. If patients deteriorate despite adequate anticoagulation and other causes of deterioration have been ruled out, thrombolysis may be a therapeutic option in selected cases, possibly in those without intracranial hemorrhage

Heparin Therapy

Dose-adjusted intravenous heparin treatment should be started immediately with a bolus of 3,000–5,000 IU after the diagnosis, even if a hemorrhagic infarct is present. Continuous treatment using an intravenous infusion system is started with 1,000–1,200 IU per hour, followed by an increase of 100–200 IU per hour every 6–8 h until aPTT is doubled. Alternatively, LMWH (e.g. nadroparin in a dose of 180 antifactor Xa U/kg/24 h administered by two

subcutaneous injections daily) can be given particularly in uncomplicated cases. Heparin therapy should be continued until remission of the acute stage of the disease (normalizing level of consciousness or remission of mental confusion, improvement of headache and focal neurological deficits).

Oral AC

After the acute stage, therapy is switched over to oral anticoagulants. Dose should be adjusted to international normalized ratio (INR) value with a target INR of 2.0–3.0. Effective anticoagulation must be ensured during adjustment of oral anticoagulants (warfarin or acenocoumerol). If CVT occurs during pregnancy, oral anticoagulants should be avoided due to its possible teratogenic effect and ability to pass the placenta. Oral anticoagulants with a target INR of 2.0–3.0 may be given for 3 months if CVT was secondary to a transient (reversible) risk factor and for 6–12 months if it was idiopathic.

Treatment of Seizures

The prophylactic use of antiepileptic drug in all patients with CVT is controversial. Whereas some authors recommend prophylactic treatment because of the high incidence of seizures (and series of seizures or even status epilepticus)

and their possible detrimental effects on the metabolic situation during the acute phase of the disease, others restrict the use of anticonvulsants to patients with seizures. Although data are insufficient to give recommendations, these findings suggest that prophylactic treatment with antiepileptic drug may be a therapeutic option for those patients, whereas it is not warranted when there are no focal neurological deficits and no focal parenchymal lesions on brain scan (e.g. patients with isolated intracranial hypertension). Prolonged treatment with antiepileptic drug for 1 year may be reasonable for patients with early seizures and hemorrhagic lesions on admission brain scan, whereas in patients without these risk factors AED therapy may be tapered off gradually after the acute stage

Treatment of Elevated ICP

Although brain swelling is observed in about 50% of all patients with CVT on CT, minor brain edema needs no other treatment than oral anticoagulants which improves the venous outflow sufficiently to reduce ICP in most patients. In patients with isolated intracranial hypertension and threatened vision, a lumbar puncture with sufficient CSF removal to obtain a normal closing pressure should be performed before starting oral anticoagulants 24 h after the puncture. There are no controlled data but acetazolamide may be considered in patients with persistent papilledema. Antiedematous treatment is necessary in only 20% of patients and should be carried out according to general principles of therapy of

raised ICP (head elevation at about 30º , hyperventilation with a target PaCO2 pressure of 30–35mm Hg, intravenous application of osmotic diuretics). However, osmotic substances might be harmful in venous outflow obstruction since they are not as quickly eliminated from the intracerebral circulation as in other conditions. Steroids can not be generally recommended for treatment of elevated ICP since their efficacy is unproven and they may be harmful through their promotion of the thrombotic process. **Treatment of Septic CVT**

Septic CVST almost always occurs in patients with bacterial cranial infections. Treatment includes the early administration of systemic antibiotics, surgical removal of the infectious focus and the use of oral anticoagulants. Antibiotics should be chosen according to the bacteria found after surgical removal, in the CSF, blood samples or in smear examinations. Treatment should be started with antibiotics which are highly effective against bacteria commonly found in infections of the face, neck or ear. The effect of heparin in septic CVT has not been systematically investigated but most authors favor the use of heparin.

Outcome

Even though it was previously thought that CVT was often leading to death, it was suggested already in 1953 that without additional morbid factors, many patients with intracranial venous thrombosis may be capable of recovery. There are several studies of long-term survival after CVT. However, only four of these studies contain more than 100 patients, and the International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT) is by far the largest study. The majority of deaths after CVT occur during the first days and months after onset of the disease. In the ISCVT, 4.3% were dead at discharge, 6.8% after 6 months and 8.3% at last follow-up (which took place after a median of 16 months in that report). Death after the acute phase may often be caused by underlying serious disease, e.g. cancer. Focal deficits and cancer in the acute phase are independent predictors of dependence or death at 3 years. However, even though most patients with CVT survive, no less than 3/4 of these survivors have been reported to have some residual symptoms. The prognosis after CVT during pregnancy and puerperium is probably better than after CVT from other causes.

AIMS AND OBJECTIVES

1. To study the demographic profile of patients admitted with diagnosis of CVT at Government Rajaji hospital during the period from January 2007 to March 2009
2. To study the clinical presentation of CVT among puerperal CVT, non puerperal female patients with CVT and male patients with CVT
3. To investigate this patient groups with CT/ MRI & MRV and evaluate the radiological findings & compare among the three groups
4. To assess the etiology, risk factors associated with CVT among puerperal CVT, non puerperal female patients with CVT and male patients with CVT
5. To assess the short term outcome among the three groups

MATERIALS AND METHODS

The study design was prospective one involving patients admitted in Medicine, Obstetrics and Gynaecology, Neuromedicine and all other wards with cerebral venous sinus thrombosis

Patients admitted and diagnosed as having cerebral venous sinus thrombosis based on CT/ MRI & MRV were included study group.

Methodology

1. Symptoms were recorded in a predesigned proforma one allotted for each patient.
2. Place of delivery and mode of delivery natural or LSCS were recorded.
3. Detailed history about risk factors for CVT enquired.
4. Detailed general examination and neurological examination about the presence of papilledema, focal deficits done and documented & compared among three groups.
5. CT / MRI & MRV findings and etiology for the cause of CVT done and entered in the proforma.
6. Short term outcome analyzed and compared among three groups.

RESULTS

Table -1

Age & Sex distribution

Age group (in years)	Puerperal CVT No = 44	Non puerperal female CVT No = 16	Male CVT No = 15
10 – 20	4 (9.1%)	3 (18.75%)	2 (20%)
20 – 30	38 (86.36%)	3(18.75%)	5(33.33%)
30 – 40	2 (4.54%)	7 (43.75%)	4(26.66%)
40 – 50		1 (6.25%)	1 (6.66%)
50 – 60		2(12.5%)	2(13.33%)
> 60 yrs		-	-

Diagram showing the percentage of Puerperal, Non puerperal & Male CVT

Puerperal - 44 (59%)

Non puerperal females - 16 (21%)

Male - 15 (20%)

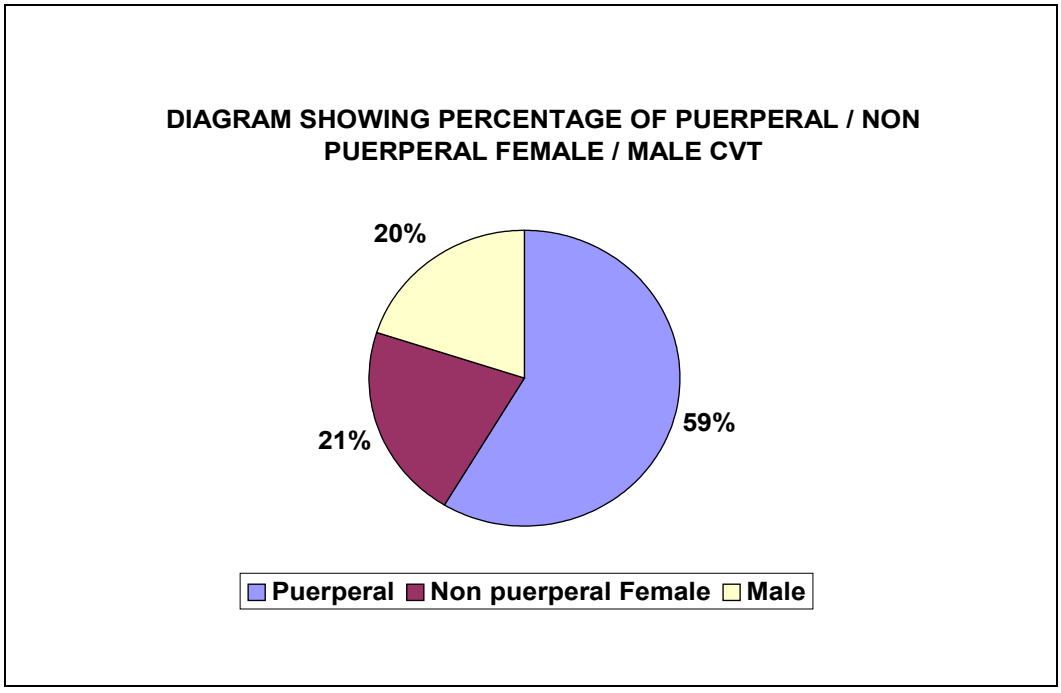

Table – 2

Symptoms of cerebral venous thrombosis

No.	Symptom	Puerperal CVT No = 44	Non puerperal female CVT No = 16	Male CVT No = 15
1.	Headache	41 (93.18%)	14 (87.5%)	15 (100%)
2.	Vomiting	31 (70.45%)	6 (39.5%)	6 (90%)
3.	Seizures – Total	29 (65.90%)	6 (39.5%)	7 (46.66%)
	Focal	4 (9.09%)	1 (6.25%)	
	Focal – GTCS	2 (4.5%)	2 (12.5%)	1 (6.66%)
	GTCS	19 (43.18%)	3(18.95%)	5 (33.33%)
	EPC	3(6.81%)	-	-
	Status epilepticus	-	-	1 (6.66%)
4.	Altered sensorium	3(6.81%)	3(18.75%)	2(13.33%)
5.	Focal neurological symptoms	29 (65.90%)	3(18.75%)	1 (6.66%)
	Right hemiplegia	13(29.59%)	2 (12.5%)	-
	Left hemiplegia	13(29.59%)	1 (6.25%)	1(6.66)%
	Monoplegia	1 (2.27%)		
	Aphasia	2(4.54%)	1(6.25%)	-
	Hemianopia	1 (2.29%)	-	-
6	Fever	3 (6.80%)	1 (6.25%)	1(6.66%)
7.	Diplopia	-	-	1 (6.66%)

Table – 3

Internal between Delivery – Onset of symptoms of puerperal CVT

Post partum day	Total = 44 No (%)
< 7 days	8 (18.18%)
7 – 9 days	9 (20.97%)
10 – 14 days	22 (50%)

15 – 21 days	5 (11.36%)
> 21 days	-

Table – 4

Signs in patients with cerebral venous thrombosis

No.	Signs	T=44 Puerperal CVT	T=16 Non puerperal	T=15 Male CVT
1.	Altered sensorium	3 (6.81%)	3(18.75%)	2(13.33%)
2.	Stupor / Coma	-	1 (6.25%)	-
3.	Ophthalmoplegia	1(2.27%)	1(6.25%)	1(6.66%)
4.	Papilledema	31(70.45%)	12(75%)	12(80.1%)
5.	Proptosis / chemosis	1 (2.29%)	1(6.25%)	-
6.	Aphasia	2(4.54%)	1 (6.25%)	-
7.	Focal neurological deficits	29 (65.90%)	3 (18.75%)	1 (6.66%)
8.	Meningeal signs	2 (4.54%)	-	1 (6.66%)

Table – 5

Presentation of Cerebral venous thrombosis

Duration	Puerperal CVT No = 44	Non puerperal Female No = 16	Male CVT No = 15
Acute < 48 hrs	7(15.9%)	2(12.5%)	1(6.66%)
Sub acute ((48hrs – 30 days)	37(84.09%)	12(75%)	9(60%)
Chronic - > 30 days	-	2(12.5%)	5(33.33%)

Diagram – 2 Association between papilledema and CVT

Puerperal CVT - 31(70.45%)

Non puerperal female CVT - 12(75%)

Male CVT - 12(80%)

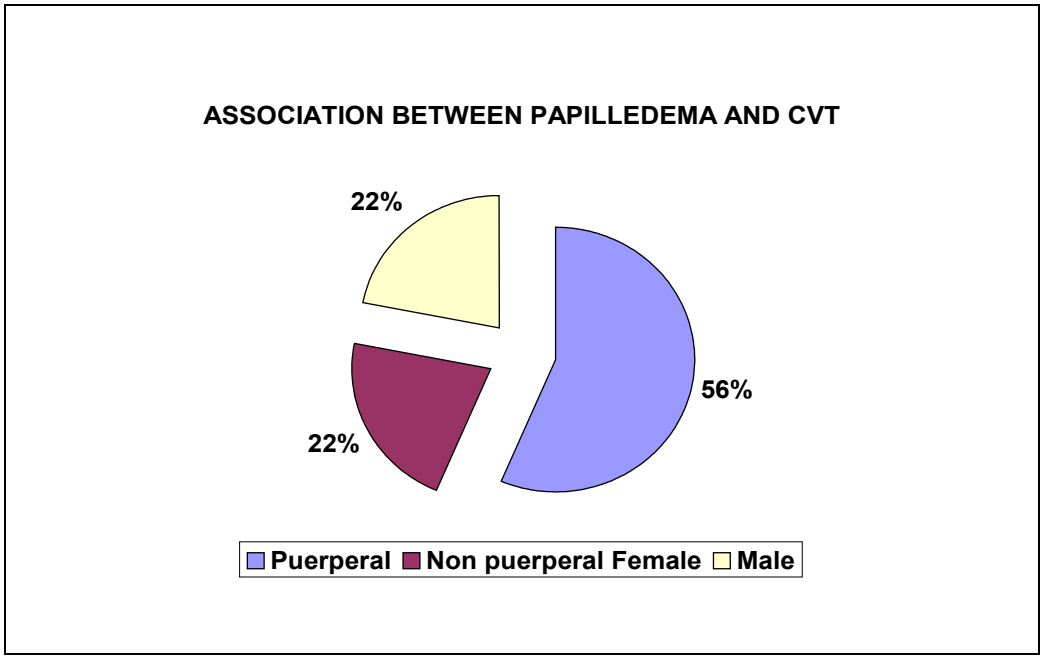

Table – 6

Investigational profile in patients with CVT

S.No.	Parameter	Puerperal CVT No = 44	NP female No = 16	Male No = 15
1.	Hb			
	Hb < 5 gm	-	-	-
	5 - 8 gm	32(72.72%)	2(12.5%)	1(6.66%)
	> 8 gm	12(27.27%)	14(87.5%)	14(93.33)
2.	Positive ANA & dsDNA	2(4.54%)	2(12.5%)	-
3	Positive APLA	-	2(12.5%)	-

4.	Low serum homocysteine	-	-	4(26.6%)
5.	Abnormal coagulation factors (proteins C, S,AT III)	NA	3(18.75%)	4(26.6%)

Table – 7

Table showing CT signs in patients CVT

Parameter	Puerperal CVT T = 44	Non puerperal CVT T = 16	Male CVT T=15
Direct Signs	16(36.36%)	11(68.75%)	7(46.66%)
Empty delta	5(11.36%)	6(37.5%)	2(13.33%)
Dense triangle	10(22.72%)	5(31.25%)	5(33.33%)
Cord sign	1(2.27%)	-	-
Indirect signs	28(63.63%)	5(31.25%)	9(60.00%)
Hemorrhagic infarct	24(54.54%)	4(25%)	8(53.33%)
Non hemorrhagic infarcts	4(9.09%)	-	-

Both	-	1(6.25%)	-
Tentorial / falx enhancement	5(11.36%)	2(12.5%)	4(26.66%)
Gyral enhancement	-	-	-
Squashing of ventricles	8(18.18%)	2(12.5%)	4(26.66%)
SAH	1(2.27%)	-	-
Midline shift	3(6.80%)	-	-
Normal CT	7(15.90%)	3(18.75%)	2(13.33%)
Thalamic hypodensity	-	1 (6.25%)	1 (6.25%)

Table – 8

Table showing sinus involved by MRV (more than one sinus involved in single patient)

Sinus Involved	Puerperal CVT No = 44	Non puerperal female CVT No = 16	Male CVT No = 15
SSS	40 (90.9%)	13 (81.25%)	11 (73.33%)
LS	33 (75.06%)	14 (87.5%)	9 (60.0%)
SS	6 (13.63%)	2 (12.5%)	1 (6.66%)
Jugular vein	1 (2.27%)	-	-
Deep veins	1 (2.27%)	1 (6.25%)	
Cavernous sinus	1 (2.27%)	1 (6.25%)	-
Anastamotic veins	2 (4.53%)	-	4 (26.66%)

Table - 9

MRI signs in CVT

Sign	Puerperal CVT group No = 44	Non puerperal female CVT group No = 16	Male CVT group No = 15
Hemorrhagic infarction	32 (72.72)	5 (31.25%)	8 (53.33%)
Frontal	12 (27.27)	1 (6.25%)	2 (13.33%)
Parietal	16 (34.09%)	4 (25%)	3 (20%)
Temporal	4 (9.09%)	-	4 (26.66%)
Occipital	1 (2.27%)	-	-
Thalamic	1 (2.27%)	1 (6.25%)	-
Non hemorrhagic infarction	6 (13.63%)	1 (6.25%)	1 (6.66%)
Frontal			
Parietal	4 (9.09%)	1 (6.25%)	1 (6.66%)
Temporal	2 (4.54%)	-	-
Occipital	-	-	-
Thalamic	-	-	-
	-	-	-
Both	-	1 (6.25%)	-

More than one location (Hemorrhagic infarction)	2 (4.54%)	-	1 (6.66%)

Table – 10

Etiological profile in patients with CVT

S.No.	Etiology	Puerperal CVT	Non puerperal Female CVT	Male CVT
1.	Post partum related	44	NA	NA
2.	Nephrotic syndrome	-	-	1(6.6%)
3.	Post chicken pox	-	-	1(6.6%)
4.	OCP use	-	2(12.5%)	-
5.	CSOM	-	-	1(6.6%)
6.	Cancer	-	1(6.25%)	1(6.6%)
7.	Head injury	-	1(6.25%)	-
8.	Connective tissue related	1(2.27)%	4(25%)	-
9.	Inherited coagulation disorders	NA	3(18.75%)	4(26.6%)

Table - 11

Outcome of CVT

Group	Puerperal CVT	Non puerperal Female CVT	Male CVT
Good Recovery	37 (84.09%)	14 (87.15%)	15 (100%)
Residual Neurological Deficit	6 (13.6%)	2 (12.5%)	-
Death	1 (1.33%)	-	-

DISCUSSION

Government Rajaji Hospital, Madurai is the only tertiary referral care hospital located in Madurai district. Various cases have been referred from Government sector hospitals like PHCs, Taluk hospitals, District head quarters hospitals, ESI hospital and many private hospital of not only from Madurai but also from near by districts.

The inpatients census crosses one lakh admissions per year from all departments.

Our prospective study of CVT patients from January 2007 to March 2009 included 75 patients. It constituted 0.59% admissions in one year (2007).

DEMOGRAPHY (Table -1)

Of the total CVT cases males constituted 20% of the cases, and remaining were females. This female preponderance 4:1 was largely due to high number of postpartum or other obstetric cause related CVT patients (80% of total). This data was consistent with previous Indian studies viz. Bansal et al[17] (1980), Srinivasan et al[12] (1983), Nagaraja et al[2] (1989). High proportion of postpartum CVT was also observed by Cantu et al[7] (1996) from Mexico. They recorded 67 out of 113 consecutive patients of CVT temporarily related to postpartum period (60.1%).

This high proportion of CVT cases not replicated in some other studies viz. Deschiens et al[53] (1996) and Daif et al[28] (1995). The possible explanation may be that the etiological factors as well as clinical profile of CVT is different in India and other developing countries where still a large number of post partum CVT are seen compared to west. Age of occurrence of CVT varied from 10 years to 60 years and more than (61.3%) (as per table No.1) fell in the 3rd decade. Ten patients are from second decade (13.33%). The mean age of all patients was 26.64 years (range 15-60 years) similar to earlier studies from India (Nagaraja et al[2] (1989). However it was significantly lower in puerperal CVT group i.e. (22.79) compared to male patients with CVT and female patients of non puerperal CVT where it was (31.53) and (32.62) respectively. Similar observation was made by Cantu et al from Mexico in his series where average age of obstetric case related CVT was significantly lower (twenty versus thirty six years). Majority of the post partum CVT in this study (fifty percent) presented ten to fourteen days consistent with other studies (Srinivasan et al[12] 1983, Nagaraja et al[2] 1987).

MODE OF PRESENTATION (Table-5)

Among the puerperal CVT, onset was acute (less than two days) in 15.9%. Cantu et al observed that evolution of symptoms was significantly more rapid in puerperal than non puerperal group of patients this in contrast to this study only 15.9% presented acutely. More common was the sub acute (84.09%) presentation

in our study. Among non puerperal CVT, sub acute presentation predominated the clinical feature (75%) of total cases. Among the male CVT presentation was sub acute (60%) and chronic (33.33%) cases. (Table no 5)

MAJOR CLASSES OF CVT (Diagram No.1)

Of the total CVT in our study, puerperal CVT constituted 58.66%, female non puerperal CVT constituted 21.33% and males 20% which is comparable to ISCVT study (Jose M Ferro et al[51] 2003) in which males constituted 25.5% and females (puerperal & non puerperal) accounted for 74.5 % .

CLINICAL PROFILE (Table-2&4)

Head ache is the major complaint in all classes of CVT which constituted 93.18%, 87.5%, 100% of cases in puerperal CVT, non puerperal CVT and male CVT respectively. This in contrast to the various Indian series where the patients presenting with headache was less (48% in Bansal et al study[17] 1980, 24% in Srinivasan et al[12] study 1983, 70.8% in Nagaraja et al [2]1989). Among the puerperal CVT group focal deficits, seizures predominant the picture (65.9% and 65.9%). Among non puerperal CVT, seizures (37.5%) and altered sensorium (18.75%) predominate the clinical feature. Among male CVT patients, GTCS alone (33.33%) dominated the clinical picture and focal neurological deficits occured only in 6.66%. This is in comparison with various Indian studies [17,2,12] of CVT. Seizures occurred in 65.90 % of puerperal CVT as compared to 37.5% of

patients in non puerperal group and 46.66 % patients in male CVT group. Aphasia was present in 2 patients (4.54%) in puerperal group and one patient (6.25%) in non puerperal group. This in contrast to the study by Cantu et al (1993) 25.3% of patients in puerperal and 21.7 % patients in non puerperal group had aphasia.

Of the puerperal CVT mean day of occurrence of CVT was 12 ± 2 days which in comparison with Indian studies (Srinivasan et al[12] 1983, Nagaraja et al[2] 1987).

Examination findings revealed papilledema in 70.45% of puerperal CVT, 75% of non puerperal CVT, 80% male CVT which in contrast to study by Bansal et al [17] and Nagaraja et al in which papilledema was present only in 35% and 58% respectively. High incidence of papilledema in this study possibly due to the more sub acute presentation of our patients compared to the above mentioned studies.

Focal neurological deficits like hemiplegia, monoplegia occurred in 61.45 % patients with puerperal CVT, 18.75 % patients with non puerperal female group and 6.66% patients with male CVT. This is in contrast to the study by Cantu et al (1993) who compared the neurological findings in puerperal and non puerperal group. In his study focal motor signs were present in 77.6% in puerperal group and 76% patients in non puerperal group.

Altered sensorium occurred in 18.75% of non puerperal CVT and 6.81% of puerperal CVT and 13.33% of patients with male CVT. Meningeal signs present in

4.5% of puerperal CVT, 6.66% of patients with male CVT which in contrast to various Indian series where the percentage of patients presenting with altered sensorium and meningeal signs are around forty to fifty eight percent.

Neurological findings in Puerperal and non puerperal CVT compared with Cantu et al study

	OUR STUDY				Study by Cantu et al 1993			
Findings	Puerperal		Non puerperal		Puerperal		Non puerperal	
	N	%	N	%	N	%	N	%
Headache	41	93.18	14	87.5	59	88	32	69.5
Focal signs	29	65.9	3	18.75	53	79.1	35	76
Motor					52	77.6	33	71.7
Sensory					25	37.3	13	28.2
Aphasia	2	4.54	1	6.25	17	25.3	10	21.7
Disorder of consciousness	3	6.81	3	18.75	42	62.6	27	58.6
Somnolence	-	-	-	-	24	35.8	9	19.5
Stupor/coma	-	-	1	6.25	16	23.9	15	32.6
Confusion	-	-	-	-	2	2.9	3	6.5
Seizures	29	65.90	6	37.5	40	59.7	29	68.0
Generalized	19	43.18	3	18.75	18	26.9	16	34.8
Focal	4	9.09	1	6.25	22	32.8	13	28.2
Bilateral pyramidal signs	-	-	-	-	28	41.7	18	19.1
Papilledema	31	70.45	12	75	27	40.2	24	52.1
Nuchal rigidity	2	4.54	-	-	22	32.8	12	26.0
Isolated intracranial hypertension	-	-	-	-	5	7.4	8	17.3

BLOOD INVESTIGATIONS (Table -6)

Anemia of moderate degree (5 to 8 gm) was present in 72.72% of puerperal

CVT but only in 12.5% and 6.6% of non puerperal and male CVT. This high percentage of anemia has been reported from various Indian studies[2, 17, 12.] Whether this reflects the anemia in the general population reflecting more severely in the puerperal group is not known. Investigations like ANA, dsDNA, APLA and coagulation profile were done only in limited number of patients which may be related to cost and availability factors. Of the patients subjected for ANA, dsDNA only 4.54% & 12.5 % of puerperal, non puerperal CVT patients were positive. Antiphospholipid antibodies were positive in only two patients with non puerperal CVT. None in the male CVT group was positive. High serum homocysteine was present in 26.66 % male patients with CVT. Evaluation for abnormal coagulation factors like Protein C, Protein S and AT III deficiency done and positive in 18.75% and 26.66 % of patients in non puerperal and male CVT group.

NEURO IMAGING – CT Scan (Table-7)

Of the total CVT patients 98.66 % of patients underwent CT, 100 % of patients underwent MRI. Of all patients subjected to CT direct signs of present in 45.33 % of patients. This in contrast to the study Cantu et al (1993) in which direct signs of CVT present only in 32.68%. Indirect signs present in 56% of patients with CVT in our study. This in contrast to study by Cantu 72.7% had indirect signs of CVT like hemorrhagic infarction, non hemorrhagic infarction and hematoma. In a study of 68 patients by Nagaraja et al (1989) 22 had direct signs of

CVT and 59 had evidence of infarction. Normal CT scans in 16 % of total patients in our study group. This in contrast with Cantu et al (1993) study in which normal CT scan was present only in 9.47%. Among direct signs empty delta signs present in 17.33% of patients, dense triangle sign in 26.66% patients & cord signs in 1.33% of patients. Dense triangle sign in puerperal group (22.72%), empty delta sign in non puerperal group (37.5%) and male CVT (33.33%) were the commonest findings. This in contrast to a study by Cantu et al (1993) cord sign and empty delta signs were present in 32 out of 95 patients (33.68%). In a study of 65 patients with CVT by Nagaraja et al (1989) cord sign was present in 14 out of 65 patients (21.53%) but in our study it was present only in 1.33% of patients. Among indirect signs hemorrhagic infarct and non hemorrhagic infarct was present in 48% and 6.66% of patients. Hemorrhagic infarct present in parietal region in 37%, frontal region 25.3%, temporal region 8 % and occipital region 1.33%, of patients. Bilateral thalamic hypo density was present in 2.66% patients suggesting deep veins involvement. Squashing of ventricles seen in 18.66% of patients. Subarachnoid hemorrhage occurred in only in one patient.

Comparison of CT findings in various studies

CT finding	Present study No = 75	Nagaraja et al 1989. n = 65	Cantu et al 1996. n = 95

Infarction	42	59	67
Cord sign	1	14	32 (cord + empty delta sign)
Empty delta	13	8	See above
Normal	12	1	9

MRI & MRV (Table – 8 & 9)

Venous sinus involved in our study compared with others studies as follows.

Sinus Involved	Present study No = 75(%)	ISCVT study, Jose M Ferro et al 2003. No = 624(%)
SSS	64(85.33%)	313(62%)
LS	56(74.66%)	536(85.9%)
SS	9(12%)	-
JUGULAR VEIN	1(1.33%)	74(11.9%)
Cavernous sinus	2(2.66%)	74(11.9%)
Deep veins	2(2.66)	68(10.9%)
Anastamotic veins	6(8%)	-

In our study as compared with ISCVT study had high percentage of

involvement of SSS and lower incidence of LS, jugular vein, deep vein thrombosis. In our study the SSS thrombosis was 90.90% in puerperal group as compared with 81.25%, 73.32% of patients in non puerperal female and male CVT. LS is involved only in 60 % of male patients with CVT. Vein of Labbe thrombosis with hemorrhage in the temporoparietal region in six of study patients (2 in puerperal, 4 in male). One of our young patients had evidence of cerebellar tuberculoma in association with CVT.

Among the parenchymal lesions in MRI, hemorrhagic infarction predominated in all three groups. 72.72% of puerperal CVT group, 31.25% in non puerperal group and 53.53 % in male CVT group had hemorrhagic infarction. In our study 44% of patients had hemorrhagic infarction in parietal region and 20% in frontal region. Non hemorrhagic infarction was present only in 10.66% of patients of total CVT in our study. This in contrast to a study by Cantu et al (1993) in which hemorrhagic infarction accounted for 52.6% in puerperal CVT group and 55% in non puerperal CVT group.

ETIOLOGY (Table – 10)

Out of total 75 patients of CVT, venous thrombosis related to pregnancy was in 58.66%. In non puerperal female CVT group, CVT was related to intake of OCP in 12.5%. one (2.27%) patient & three (18.75) patients in nonpuerperal female group had cancer and connected tissue disorder respectively as a risk factor

for CVT. In male patients, CVT was related to nephrotic syndrome in one patient (6.66%), exanthematous fever (chicken pox) in one patient (6.66%), CSOM in one patient (6.66%), and cancer in one patient (6.66%). Head injury as risk factors of CVT in only one patient in non puerperal group. Inherited coagulation disorders were the risk factors of CVT in 9.33% of patient with total CVT. This very low percentage was related to less number of patients underwent these investigation because of cost factors. Alcoholism was a major risk factor for male patients with CVT in our study (53.33%)

OUTCOME (Table – 11)

13.63% patients in puerperal group and 12.5% patients in non puerperal group had residual neurological deficit & none in male CVT group. One patient (2.27%) died in puerperal CVT group. No death occurred in non puerperal CVT and male CVT group.

SUMMARY & CONCLUSION

1. Over a period of 27 months, 75 patients of cerebral venous thrombosis were studied. Almost more than one half (58.66%) of patients were post partum CVT, data consistent with most Indian studies.
2. CVT still remains a disease of young adults. In this study, 61.33% of patients were below 30 years of age.
3. Mean age was significantly lower in puerperal group compared to non puerperal group.
4. Majority of post partum CVT patients presented with in 10-14 days after delivery.
5. Majority in our study presented subacutly (48 hrs – 30 days). There was no significant difference between puerperal and nonpuerperal group (non puerperal female and male CVT).
6. Headache (70/75), Seizures (42/75), and focal neurological deficits (33/75) were the major clinical features noted. There was no difference between puerperal, non puerperal female and male CVT in terms of frequency of these symptoms.
7. Haemoglobin value of less than 8 gm in 46.66% of total CVT. In

puerperal group, this number was high (72.72%) compared to non puerperal female CVT and male CVT.

8. Cerebral infarction was the most common abnormality noted on the CT scan (56%) which was haemorrhagic in most of the cases. Deep seated venous thrombosis noted only in 2.66% (2/75) of patients. 1 in puerperal group and 1 in non puerperal female CVT group.

9. On MRI most common sinus involved in all the three groups was SSS (67/75) 89.33%. The next common sinus involved was LS (74.66%). There was no significant difference in puerperal, non puerperal and male CVT group in the frequency of SSS and LS involvement.

10. In MRI haemorrhagic infarction was present in 72.72%, 31.25%, 53.33% of patients in puerperal, non puerperal female and male CVT group respectively. Haemorrhagic infarction is most commonly present in the parietal region in 23/75 patients. Frontal haemorrhagic region was present less commonly (15/75 patients). One patient had occipital haemorrhagic infarct in puerperal group. Haemorrhagic infarction in more than one location was present in 4% (3/75) total patients with CVT.

11. 44 out of 75 patients the venous thrombosis was related to puerperium. Evaluating the risk factors for CVT in non puerperal group showed association with OCP intake in 2 patients, Related to cancer CVT in one,

related to SLE in three (18.75%). Head injury was the risk factor for CVT in one non puerperal CVT patient. Among male patients with CVT nephritic syndrome, chicken pox, CSOM, Cancer each one was associated in CVT in single patient. Among male CVT group alcoholism was the major risk factor (53.33%).

12. Out come was better in male patients with CVT since no death or residual neurological deficit was reported. 2 out of 16 patients in nonpuerperal group had residual neurological deficit. In puerperal group, 1 patient died and 6 had residual neurological deficit.

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ABBREVIATIONS

CVT	Cerebral Venous Thrombosis
SSS	Superior Sagittal Sinus
LS	Lateral Sinus
SS	Sagittal Sinus
GTCS	Generalised Tonic clonic Seizures
EPC	Epilepsia Partialis Continua
CT	Computed Tomography
MRI	Magnetic Resonance Imaging
MRV	Magnetic Resonance venography
SAH	Subarachnoid haemorrhage
ICH	Intra Cerebral Haemorrhage
LMWH	Low molecular weight heparin
ANA	Anti Nuclear Anti body
dsDNA	Double standard Deoxy ribo Nucleic Acid
APLA	Anti Phospholipid Antibody
CSOM	Chronic Suppurative Otitis Media
NA	Not Applicable

PROFORMA

S.No: D.O.A: D. of Delivery:

Name Age/ Sex D.O.D:

Para: Gravida: Interval between delivery & first symptom:

SYMPTOMS: Interval between first symptom & admission:

Headache: Duration: Site:

Type:

Vomiting :

Seizures: Focal: Generalized: Focal – generalized: EPC:

Status epilepticus:

Consciousness: Normal / Altered: (Duration):

Focal deficits: Motor: Hemiparesis / monoparesis / paraparesis / tetraplegia

Sensory: Visual:

Mental symptoms: Normal / confused / apathetic / others

Fever:

Course since the onset of symptoms: static / improving / progressing

Delivery: Home/ PHC/ Nursing home/ GRH Normal / LSCS

PAST HISTORY :

CVT: DM: HT: Seizures: Hematological disorder:

Ear discharge: OCP: Renal disease: Alcohol intake:

Exanthematous fever: Cancer: Recent surgery:

Bloody diarrhea: Vasculitis (rash, arthritis):

EXAMINATION:

Pulse: BP: Temp:

Dehydration: Thrombophelibitis: DVT:

Consciousness: Orientation: Behaviour:

Language:

Meningeal signs: Visual acuity: Eye movements:

Pupils: Fundus:

Facial weakness Deafness Dysphagia

Motor system Weakness Power Tone

Reflexes

Sensory system

Bladder involvement

Cerebellar signs

CVS RS ABDOMEN

INVESTIGATONS:

Hb ESR: TC: Urine:

BT CT ANA: S. Homocysteine

PS APLA:

Blood sugar Urea S.Creatinine: LFT

Electrolytes CXR S.Fibrinogen

ECG Others – (Protein C & S, AT III)

CT scan: plain / contrast Normal / Abnormal

Direct signs – empty delta sign / dense triangle sign / cord sign (SSS / LS /Medullary)

Indirect signs – hemorrhagic infarct / non hemorrhagic infarct / both

Location - frontal / parietal / temporal / occipital / thalamic

Tentorial / falx enhancement

Gyral enhancement SAH

Mass effect - squashing of ventricles / midline shift / obliteration of basal cisterns

MRI & MRV:

Hemorrhagic infarct / non hemorrhagic infarct / both

Location - frontal / parietal / temporal / occipital / thalamic

Stage of sinus thrombosis

Sinus involved – SSS / LS / SS / Cavernous sinus / Deep veins / combinations

Anastamotic veins

OUTCOME:

PRESENTATION: acute / subacute / chronic Septic / Aseptic

SINUS INVOLVED: SSS / LS/ TS/ SS / Cortical vein / Deep venous system

DIAGNOSIS: Puerperal CVT / Non Peurperal CVT in Females / Male CVT

KEY TO MASTER CHART

Puerperal state

1. Puerperal 2. Non puerperal 3. Not applicable

Headache

0 – absent 1- present

Vomiting

0 – absent 1- present

Seizures

0 – absent 1- focal 2- focal – generalized

3- GTCS 4- EPC 5- Status epilepticus

Altered sensorium

0 – absent 1- drowsy 2- stupor / coma

Fever

0 – absent, 1- present

Focal neurological deficit

0 –absent 1- Rt hemiplegia 2-Lt hemiplegia

3- Monoplegia Rt 4- monoplegia Lt 5- Homonymous hemianopia

Aphasia

0 – absent 1- present

Diplopia

0 – absent 1- present

Opthalmoplegia

0 – absent, 1- present

Papilledema 0 – absent, 1- blurring of nasal margins,

2 - established Papilledema,

3 - papilledema + fundal hemorrhages

Inteval between delivery & onset of symptoms of CVT

0 - not applicable , 1- $\leq$ 7 days , 2- 7-9 days,

3 - 10-14 days, 4- 15-21 days, 5- > 21 days.

Hb(gms)

1- ≤ 5 gms 2- 5-8 gms 3- > 8 gms

Vasculitic profile

0- negative 1- positive ANA 2- positive ANA & ds DNA

3- positive APLA 4- not done

Coagulation factors

0- normal 1- low protein C 2- low protein S

3- low AT III 4- not done

CT scan findings

0- normal 1-Empty delta sign 2- dense triangle sign

3- cord sign 4-hemorrhagic infarct

5- both hemorrhagic infarct& non hemorrhagic infarct

6-tentorial / falx enhancement 7- gyral enhancement

8-squashing of ventricles 9- SAH

10- mid line shift 11- non hemorrhagic infarct 12- not done

MRI sinus involved

1- SSS 2- LS 3- SS 4- SSS+LS

5- SSS+LS+SS 6- LS+SS 7-SSS+LS+SS+Jugular vein

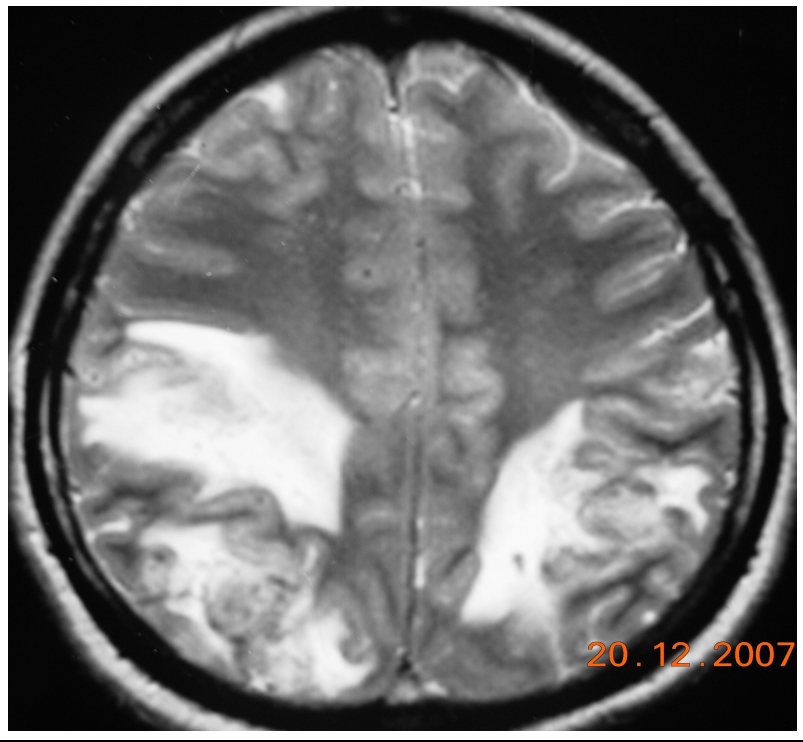
8- cavernous sinus 9- deep venous system

Out come

1- Good recovery

2- resi[illegible]

3- dea[illegible]



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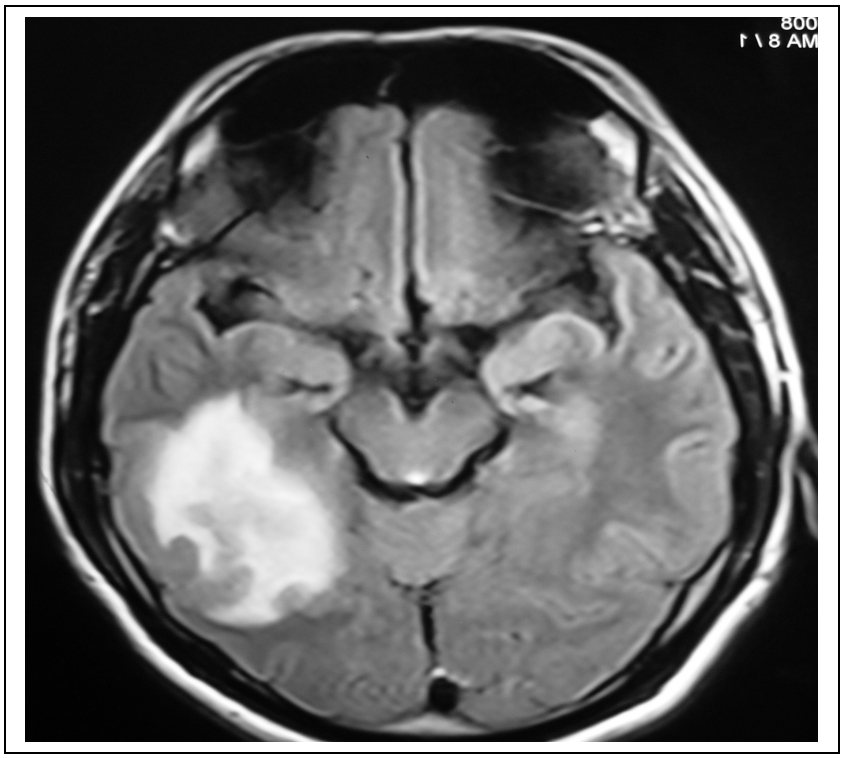
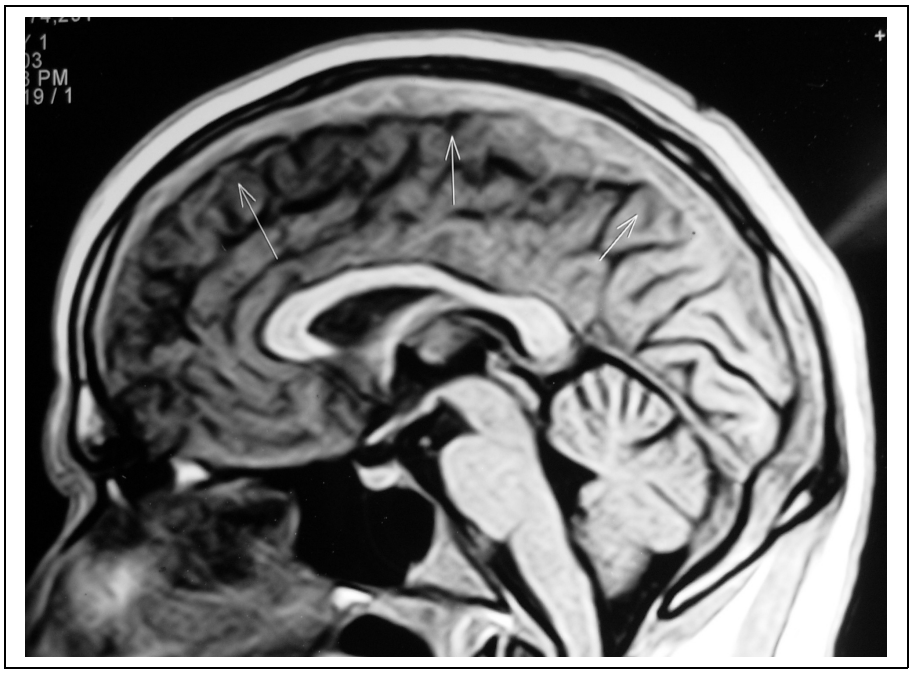
MRI BRAIN SHOWING HAEMORRAGE IN TEMPORAL REGION IN A PATIENT WITH LATERAL SINUS THROMBOSIS



MRI MID SAGITTAL SECTION SHOWING EVIDENCE OF SUPERIOR SAGITTAL SINUS THROMBOSIS



MRV SHOWING LATERAL SINUS THROMBOSIS